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Energy expenditure in the acute renal failure patient mechanically ventilated

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Abstract. Twenty mechanically ventilated patients with acute renal failure were studied on 31 occasions to determine their energy expenditure (EE) during a 2 h period before a hemodialysis. Oxygen consumption and CO_2 elimination were measured continuously with a mass spectrometer system. EE (1660 ± 48 kcal day^{-1}) was close to the total caloric intake (1682 ± 83 kcal day-1) and represented 1.19 ± 0.03 times the predicted resting energy expenditure (PREE) with large inter-individual variations (0.7–1.7 PREE). EE/PREE was higher when sepsis was present (1.31 ± 0.03 versus 1.14 ± 0.02; $p<0.05$). Glucose oxidation rate (4.35 mg kg^{-1} min^{-1}) exceeded glucose intake (2.6 mg kg^{-1} min^{-1}). Respiratory quotient was 1.02 ± 0.01. Nitrogen loss was 17.3 ± 1.7 g day^{-1} and nitrogen balance −11.9 ± 1.9 g day^{-1}. In conclusion, EE values were scattered but never exceeded 1.7 times the PREE. Sepsis increased EE. With a nutritional support covering EE, nitrogen balance remained markedly negative and a preferential utilisation of glucose and lipogenesis occurred.

Key words: Acute renal failure – Energy expenditure – Indirect calorimetry – Mass spectrometry

The energy expenditure (EE) in selected groups of critically ill [6, 9, 14], burned [17] or post-operative patients [20] has been reported to vary from 1.2 to 1.5 times the predicted resting energy expenditure (PREE). EE in acute renal failure (ARF) patients has been poorly investigated, and was found to be 1.5 [4] to 2.5 [15] times the PREE. The present study was designed (1) to reevaluate EE in ARF patients being mechanically ventilated by using a mass spectrometer system for continuous measurement of O_2 consumption ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$) and (2) to examine in these patients the relationships between EE, and sepsis, caloric intake and protein catabolic rate.

Patients and methods

Patients

Twenty hemodynamically stable, non edematous patients were studied; all had ARF requiring hemodialysis. They were mechanically ventilated in the control mode for acute respiratory failure. At the time of the study, their arterial PO_2 was above 8 kPa with a F_IO_2 less than 0.4. The mean age was 61.2 ± 2.4 years (range: 28–76). The origin of ARF was medical in 11 patients, post-surgical in 6 patients and post-traumatic in 3 patients. On 10 occasions, patients were septic (sepsis was assumed when three of the following five criteria were present: temperature above 38.5 °C for several days, leukocytosis, positive blood cultures, presence of an internal abscess, peritonitis). All patients received parenteral and/or an enteral nutritional support. No attempt was made to regulate the nutrient intake which was prescribed by the attending physician. Mean caloric intakes are listed in Table 1. For 6 h prior to the study and during the whole study time glucose was the sole nutrient and was given at a mean constant rate of 2.6 mg kg^{-1} min^{-1}; no patient was receiving insulin.

Table 1. Caloric intakes during 31 studies in 20 acute renal failure patients. Values are the means ± SEM

Caloric intake kcal day^{-1}	Glucose intake g day^{-1}	Fat intake g day^{-1}	Protein intake g day^{-1}
1682 ± 83	257 ± 18	55 ± 3	34 ± 3

Protocol

On 31 occasions, $\dot{V}O_2$ and $\dot{V}CO_2$ were measured during four consecutive 30 min periods, before a hemodialysis session. We used a mass spectrometer system (Perkin Elmer MGA 1100) which provides a measurement of $\dot{V}O_2$ and $\dot{V}CO_2$ each 200 s. The mean of the nine consecutive values was considered as the $\dot{V}O_2$ and the $\dot{V}CO_2$ for each 30 min period. Details of the mass spectrometer system have been presented in a previous report [2]. The system can be briefly described as follows: gas samples were drawn from the Y piece of the patient's breathing circuit to a mass spectrometer and analyzed for inspired O_2 concentration and CO_2 wave form recognition. The latter analysis allowed rejection of artifactual cycles, e. g. due to coughing or tracheal suction. Then, expired gas was sampled from the outlet of a mixing chamber for the measurement of mixed expired O_2 and CO_2 concentrations. Expired flow was measured by a pneumotachometer (Gould). All the signals were collected by a microcomputer (Kontron) progammed to reject artifactual respiratory sequences and to compute $\dot{V}O_2$ and $\dot{V}CO_2$.

Calculations (see Appendix)

PREE [Eq. (1)] was calculated according to the reevaluated Harris-Benedict equation [16], using the actual body weight.

EE [Eq. (2)] and the apparent glucose oxidation rate (GOR) [Eq. (3)] were calculated according to the post absorptive formulae given by Bursztein et al. [5].

Protein catabolic rate was calculated from the rate of urea nitrogen production during 24 consecutive h including the 8 h of the study period. During this period, patients had no hemodialysis. Urea nitrogen production value [Eq. (4)] was calculated as the sum of the urinary urea nitrogen loss, and the change in body urea nitrogen level as proposed by Blumenkrantz et al. [3].

The results are presented as the means ± SEM. Statistical analysis used Student's t-test for comparison of means and least-squares fit method for linear regression.

Results

Mean EE was close to the total caloric intake and represented 1.19 ± 0.03 times the PREE (Table 2). As shown in Figure 1, the ratio EE/PREE varied from 0.8 to 1.7. When sepsis was present, EE/PREE was 1.31 ± 0.03. In the absence of sepsis, EE/PREE was significantly lower (1.14 ± 0.02; $p < 0.05$). There was no correlation between EE and core temperature.

Mean glucose oxidation rate (4.35 mg kg^{-1} min^{-1}) exceeded mean glucose intake (2.6 mg kg^{-1} min^{-1}). Respiratory quotient (RQ) was above one (Table 2).

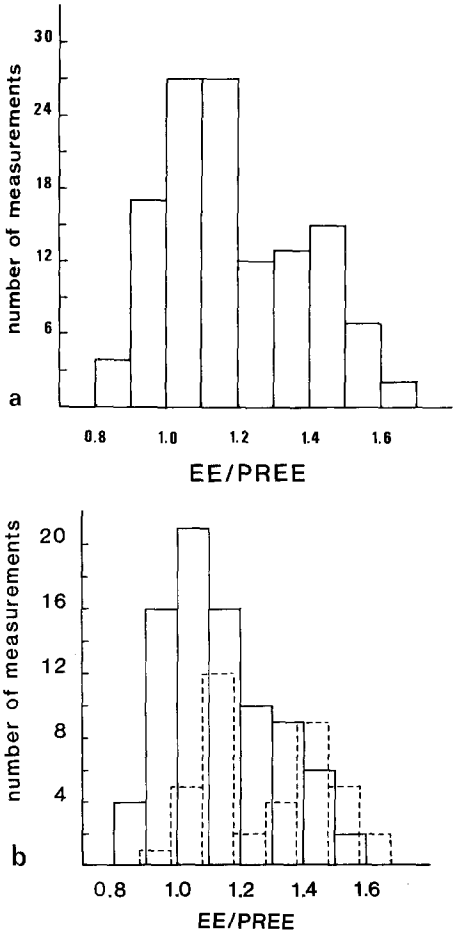

Fig. 1. Histograms of energy expenditure/predicted resting energy expenditure (EE/PREE) in acute renal failure patients. b □ = non septic; ⬚ = septic

Table 2. Energy expenditure during 31 studies in 20 acute renal failure patients. Values are the means ± SEM

$\dot{V}O_2$ ml·min^{-1}·m^2	$\dot{V}CO_2$ ml·min^{-1}·m^2	RQ	PREE kcal·day^{-1}	EE kcal·day^{-1}	Nitrogen loss g·day^{-1}	Nitrogen balance g·day^{-1}	Glucose oxidation rate		Protein catabolic rate g·day^{-1}
							g·day^{-1}	mg·kg^{-1}·min^{-1}	
135 ±4	137 ±3	1.02 ±0.01	1400 ±26	1660 ±48	17.3 ±1.7	−11.9 ±1.9	400 ±15	4.35 ±0.19	108 ±11

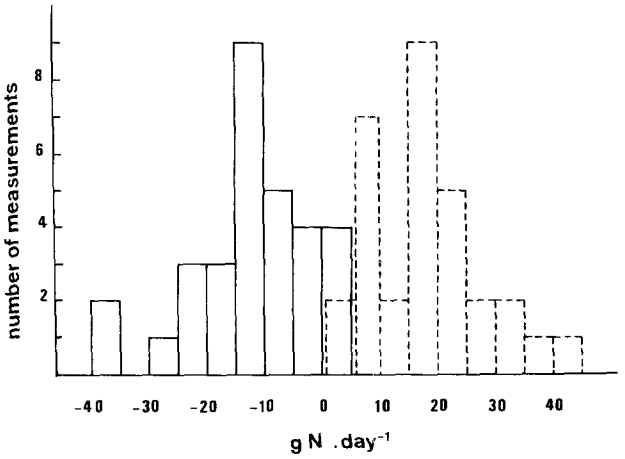

Fig. 2. Histograms of nitrogen balance and nitrogen loss in acute renal failure patients. □ = nitrogen balance; ⬚ = nitrogen loss

There was a large nitrogen loss and mean nitrogen balance was negative (Table 2). As shown in Figure 2, there was a large inter-individual variation in nitrogen balance which varied from +5 to −40 g N day^{-1}. There was no difference between septic and non septic patients.

There was no correlation between EE, nitrogen balance and caloric intake, nor between nitrogen balance and nitrogen intake.

Discussion

The evaluation of EE by indirect calorimetry requires multiple and frequent determinations of $\dot{V}O_2$ and $\dot{V}CO_2$, particularly in intensive care patients. We therefore, used a mass-spectrometer system designed to provide an accurate continuous measurement of pulmonary gas exchange during mechanical ventilation [2].

We found that mean EE was 1.19 times the PREE. This result is close to the values measured in other intensive care patients [11, 14, 17, 20] and in the three ARF patients studied by Braun et al. [4]. On the contary, Miller et al. [15], in ten ARF patients, found EE to be elevated much more some 2.5 times the PREE. They used the Douglas bag technique, a method which has the limitation of intermittent data availability.

The EE values, which we measured, were scattered. This indicates that ARF is not associated with a predictable increase in EE. In particular, the level of the protein catabolic rate is not a good indicator of the level of EE. The only factor which increased EE significantly was sepsis as found by others [7, 9] in various groups of intensive care patients.

In our study, glucose oxidation rate largely exceeded glucose intake. This indicates marked endogenous production of glucose, glycogenolysis and/or neoglucogenesis. The participation of neoglucogenesis is suggested by the markedly negative nitrogen balance. Moreover, an accelerated obligatory neoglucogenesis has been shown in chronic renal failure [12] and during sepsis [1]. A large caloric intake during ARF has been recommended [8, 10, 13, 18, 19] to prevent protein catabolism. On the basis of our results, the nutritional benefits of such a caloric load could be questionable. In fact, with a nutritional support just covering EE, we found a RQ above one, which indicates a preferential utilisation of glucose and the existence of lipogenesis. A larger caloric intake could enhance the lipogenesis without avoiding the protein catabolism. This might lead to accumulation of fat in the liver and thus to liver dysfunction.

In conclusion, EE values during ARF were scattered but never exceeded 1.7 times the PREE. With a nutritional support covering EE, nitrogen balance remained markedly negative and a preferential utilisation of glucose and a lipogenesis occurred.

Appendix

Eq. (1). Predicted resting energy expenditure (PREE). For men PREE = 88.362 + 4.799 H + 13.397 W − 6.673 A. For women PREE = 447.593 + 3.098 H + 9.247 W − 4.330 A. PREE (kcal day^{-1}); H (cm) = height; W (kg) = weight; A = age.

Eq. (2). Energy expenditure (EE). EE = 5.083 $\dot{V}O_2$ + 0.138 $\dot{V}CO_2$ − 0.128 UNA. EE (kcal day^{-1}); $\dot{V}O_2$ (l day^{-1}); $\dot{V}CO_2$ (l day^{-1}). UNA (g N day^{-1}) = rate of urea nitrogen appearance.

Eq. (3). Apparent glucose oxidation rate (GOR). GOR = 4.06 $\dot{V}CO_2$ − 2.854 $\dot{V}O_2$ + 0.095 UNA. GOR (g day^{-1})

Eq. (4). Urea nitrogen appearance (UNA). UNA = UUN + ($SUN_f - SUN_i \cdot 0.6\, W_i$) + ($W_f - W_i \cdot SUN_f$). UUN (g day^{-1}) = urinary urea nitrogen; SUN (g l^{-1}) = serum urea nitrogen; i = initial value; f = final value.

References

1. Beisel WR, Wannemacher RW (1980) Glucogenesis, ureagenesis and ketogenesis during sepsis. JPEN 4:277
2. Bertrand O, Viale JP, Annat G, Sebes F, Delafosse B, Percival C, Bui Xuan B, Motin J (1986) A mass spectrometer system for long term continuous measurement of $\dot{V}O_2$ and $\dot{V}CO_2$ during artificial ventilation. Med Biol Eng Comput 24:174
3. Blumenkrantz MJ, Kople JD, Gutman RA, Chan YK, Barbour GL, Roberts C, Shen FH, Ganghi VC, Tucker CT, Curtis FK, Coburn JW (1980) Methods for assessing nutritional status of patients with renal failure. Am J Clin Nutr 33:1567
4. Braun U, Berger C, Kunze E, Martell J, Schwarzkopf J, Trapp V, Kramer P (1985) Daily energy and nitrogen balance in acute catabolic renal failure. In: Continuous arterio-venous hemofiltration. International conference on CAVH, Aachen 1984. Karger, Basel, p 219
5. Bursztein S, Glaser P, Trichet B, Taitelman U, Nedey R (1980) Utilization of protein, carbohydrate and fat in fasting and post absorptive subjects. Am J Clin Nutr 33:998
6. Carlsson M, Thorne A, Forsberg E (1985) Energy expenditure and thermogenic effect of nutrition in long term critically ill trauma sepsis patients. 7th Congress of European Society of Parenteral and Enteral Nutrition, Munich. Abstract 0.116

7. Elwyn DM (1980) Nutritional requirements of adult surgical patients. Crit Care Med 8:9
8. Feinstein EI, Blumenkrantz MJ, Healy M, Koffler A, Silberman H, Massry SG, Kopple JD (1981) Clinical and metabolic responses to parenteral nutrition in acute renal failure. Medicine 60:124
9. Giovannini I, Boldrini G, Chiarla C, Castagneto M, Castiglioni GL (1985) Hypermetabolism and substrate utilization in sepsis and trauma. 7th Congress of European Society of Parenteral and Enteral Nutrition, Munich. Abstract 0.32
10. Grodstein GP, Blumenkrantz MJ, Kopple JD (1980) Nutritional and metabolic response to catabolic stress in uremia. Am J Clin Nutr 33:1411
11. Hunker FD, Bruton CW, Hunker EM, Durham RM, Krumdieck CL (1980) Metabolic and nutritional evaluation of patients supported with mechanical ventilation. Crit Care Med 8:628
12. Kalhan SC, Ricanati ES, Tserng KY, Savin SM (1983) Glucose turnover in chronic uremia: increased recycling with diminished oxidation of glucose. Metabolism 32:1155
13. Kosanovich JM, Dumler F, Horst M, Quandt C, Sargent JA, Levin NW (1985) Use of urea kinetics in the nutritional care of acutely ill patient. JPEN 9:165
14. Mann S, Westenskow DR, Houtchens BA (1985) Measured and predicted caloric expenditure in the acutely ill. Crit Care Med 13:173
15. Miller RL, Taylor WR, Gentry W, Day AT, Gazzaniga AB (1983) Indirect calorimetry in post operative patients with acute renal failure. Am Surg 49:494
16. Roza AM, Shizgal HM (1984) The Harris Benedict equation reevaluated: resting energy requirements and the body cell mass. Am J Clin Nutr 40:168
17. Saffle JR, Medina E, Raymond J, Westenskow D, Kravitz M Warden GD (1985) Use of indirect calorimetry in the nutritional management of burned patients. J Trauma 25:32
18. Shenkin A (1979) Monitoring the nutritional status of critically ill patients. Intensive Care Med 5:165
19. Spreiter SC, Myers BD, Swenson RS (1980) Protein energy requirements in subjects with acute renal failure receiving intermittent hemodialysis. Am J Clin Nutr 33:1433
20. Weissman C, Kemper M, Damask MC, Askanazi J, Hyman AI, Kinney JM (1985) Metabolic rate in the post-operative critical care patient (Abstract) Crit Care Med 13:280

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